PERIPHERAL OBLITERATING ARTERITIS AS A CAUSE OF TRIPLEGIA FOLLOWING HEMIPLEGIA.*

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We published in the *American Journal of the Medical Sciences* for June, 1905, a paper entitled "Peripheral Obliterating Arteritis as a cause of Triplegia following Hemiplegia, and of Paraplegia." In it we described a not very infrequent but much neglected form of palsy brought about by obliterating arteritis in the extremities affected. In this condition the leg on the non-paralyzed side becomes as powerless as the other. Both thighs become flexed on the abdomen and the calves on the thighs. There is marked rigidity and some muscular wasting, not a local atrophy picking out individual muscles but a general decrease in size. The skin is mottled, bluish-red and looks and feels thin. The final condition presents a clinical picture superficially resembling that seen when diffuse myelitis has occurred in hemiplegia, but there is not true palsy of the bladder and rectum. The condition must not be mistaken for the triplegia which sometimes occurs in syphilis as the result of multiple cerebral and spinal lesions, nor for double hemiplegia or hemiplegia plus monoplegia resulting from bilateral cerebral lesions. It slowly follows a single cerebral apoplexy, and is caused not by secondary involvement of the spinal cord but by disease of the arteries of the legs themselves. It occurs only in old people whose arteries are diseased throughout the entire body. There is no reason why it should not affect the arms as well as the legs but we have never seen such a case.

During the past winter another instance of this condition has come under our care and we now report it. The history of the case is as follows:

W. E., a white man, 80 years old, was admitted to Dr. Burr's service in the Philadelphia Hospital in March, 1904, and died in

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January, 1906. On admission to the hospital he was suffering from a partial right hemiplegia and aphasia. All that we could discover concerning the history of his illness was that he had had a stroke a few days previously. There was slight right-sided facial palsy. He could move the tongue well in all directions. There was paralysis of the right arm. Both legs were somewhat weak but weakness was greater on the right side. There was no disturbance of movement in the left arm or leg. The knee jerk on the right side was somewhat decreased, on the left it was normal. Stroking either sole caused slight flexion of all the toes followed on the right side by extension of the great toe. Sensibility was normal except in the right hand and forearm where sense of touch and pain was lost. The skin over the hands and feet was distinctly atrophic. The pupils were equal, reacted promptly to light, slowly to accommodation. All movements of the eyeballs were normal. The patient understood all that was said to him but could speak very little using only single words. He could recognize objects by sight and indicated that he knew the use of them but could rarely call their name. He could recognize objects by touch in the left hand but not in the right. He could understand short written or printed words. After some days his speech had improved a great deal and the anesthesia passed away. At the first examination there were no contractures of either leg or arm. By January, 1906, he was completely bedridden. Both thighs were flexed upon the abdomen, the knees closely pressed together, the calves flexed upon the thighs, and one foot crossed over the other. This condition slowly increased for months. It was not due to active spasm but apparently to shortening of the muscles. The right arm was also markedly contractured, the left not at all. The femoral pulse was palpable on either side, but the popliteal pulses could not be felt. Both legs were cold, the feet bluish and the toe-nails almost black. The right knee jerk could not be obtained. (This may have been either on account of the great rigidity or because of the distortion of the knee from old disease in it.) The left knee jerk was quite marked. Stroking the right sole caused drawing up of the whole leg without any movement of the toes. Stroking the left side caused no movement of any kind. There was slight Achilles jerk on the right side, none on the left. The biceps jerk was present on both sides. He had lost all power of articulation except to say "yes" and "no." He understood all simple verbal commands. Several days later he became stuporous and died shortly after.

At autopsy the brain appeared superficially normal. There was slight but not great atheroma of the arteries. There was slight enlargement of the ventricles. The popliteal arteries on both sides contained thrombi. The iliac arteries were much thickened. On cross section of the brain a small area of softening was found in the left internal capsule and another in the island of Reil. The other lesions found at necropsy were chronic interstitial nephritis, and chronic myocarditis.

Histological examination of the paracentral lobules showed much congestion within the sections. The blood vessel walls were only moderately thickened and there was slight fibrous thickening of the pia. The Betz cells, though exceedingly pigmented, appeared normal by the thionin stain. The Mallory neuroglia stain showed moderate subpial glial hypertrophy.

A series of sections of the right internal capsule showed it to be normal by the hemalum-acid-fuchsin and Weigert hematoxylin stains. On the left side a series of sections showed sclerosis in the island of Reil and an area of degeneration of fibers in the posterior limb of the internal capsule when stained by the Weigert hematoxylin method. A series of sections of the cerebral peduncles showed the left to be distinctly smaller than the right and there was an area of degeneration occupying the middle third of the left crusta, but otherwise the sections appeared normal. In the pons serial sections showed degeneration of some of the bundles of fibers of the pyramidal tract on the left side when stained by the Weigert hematoxylin method, but no degeneration on the right side. There was complete degeneration of the left pyramidal tract in the medulla, but the right pyramidal tract stained very well by the Weigert method. The small intramedullary blood vessels were very prominent, caused rather by congestion than by thickening of their walls. There was a fairly well marked subpial gliosis but no round cell infiltration about the blood vessels nor in the pia. The cells of the nuclei of the cranial nerves stained by the thionin method appeared to be in fairly good condition.

Sections from the cervical region of the spinal cord stained by the Weigert method showed an area of degeneration in the right crossed pyramidal tract, and very slightly in the left direct pyramidal tract. The Marchi method revealed recent degeneration in the same location. There was no degeneration in other parts of the section. Sections stained by the hemalum-acid-fuchsin method showed a well marked thickening of the walls of the finer blood vessels making them appear unduly prominent. There was an overgrowth of glia about the blood vessels and also a moderate subpial gliosis. The nerve cells in the anterior horns stained by the thionin method while intensely pigmented were otherwise normal. Sections from the thoracic and lumbar regions of the spinal cord do not require separate description, as the same conditions were there present as in the cervical region, except that there was no degeneration in the left direct pyramidal tract below the level of the fourth thoracic segment.

To summarize: the findings in the central nervous system were only those found in an ordinary case of right-sided hemiplegia, an area of sclerosis in the left cerebral hemisphere and the usual secondary degeneration of the pyramidal tract on that side. There was no evidence of a lesion on the opposite side. Other findings in the brain and spinal cord cannot be regarded as pathological in a man of the patient's age, as they are found in numerous persons in whom during life there are no nervous symptoms.

Sections of the popliteal nerves stained by the hemalum-acid-fuchsin method showed a marked increase of connective tissue between the nerve bundles, and to a less extent within them. There was no evidence of any inflammatory change, and by the Weigert hematoxylin method the nerve fibers stained well. There was no notable difference between the nerves from the two legs. When stained by the Marchi method there was seen, in longitudinal sections, black staining masses within the myelin not having, however, a typically degenerated appearance.

Sections of the left external iliac artery showed marked irregular thickening of the intima (endarteritis nodosa) and in one portion a hemorrhage into the intima. There was marked fibrosis of the media with calcification. Sections of the right external iliac artery showed even more thickening of the walls and constriction of the lumen than the left. The left popliteal artery was filled with a recent thrombus and its walls were greatly thickened, especially the intima. A small blood vessel seen in the same section, a branch of the popliteal, was almost completely occluded by intimal thickening. The right popliteal resembled the left.

Sections of the right tibialis posticus muscle stained by hematoxylin eosin showed a moderate increase in the connective tissue and of the connective tissue nuclei. The small intramuscular nerve fibers stained well by the Weigert hematoxylin method, as did also a nerve fiber seen within a muscle spindle. Sections of the left tibialis posticus stained by the hematoxylin-eosin appeared nearly normal. There was a slight increase in the amount of connective tissue between the muscle fasiculi without any increase of connective tissue nuclei. The muscles presented no apparent increase in sarcolemma nuclei. Stained by the Marchi method they showed distinct recent degeneration, a finely granular fatty change, present in numerous fibers throughout the sections.

In the nerves there was but a slight fibrosis not abnormal when the age of the patient is considered. The blood vessels showed intense arterio-sclerosis more marked in the smaller arteries where it produced obliteration in many. The muscles showed a distinct degenerative change by the Marchi method of staining.

It is of course well known that peripheral arterial disease may cause a palsy of the affected extremities. Many years ago Charcot showed that disease of the larger arterial trunks is the cause of intermittent claudication. It is well established that in many instances the motor disability of old age (often in senile paraplegia for example) is frequently brought about not by cerebral or spinal cord disease but by lack of muscular nutrition on account of poor blood supply through the much thickened and diseased arteries and arterioles. This was the condition found in our patient. When there has been no preceding hemiplegia the picture is simple enough but when the two are combined the result resembles superficially a severe spastic myelitis.